

Discuss (2) Improve

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Nephrotic syndrome

Triad of:

- 1. Proteinuria (> 3g/24hr) causing
- 2. Hypoalbuminaemia (< 30g/L) and
- 3. Oedema

Loss of antithrombin-III, proteins C and S and an associated rise in fibrinogen levels predispose to thrombosis. Loss of thyroxine-binding globulin lowers the total, but not free, thyroxine levels.

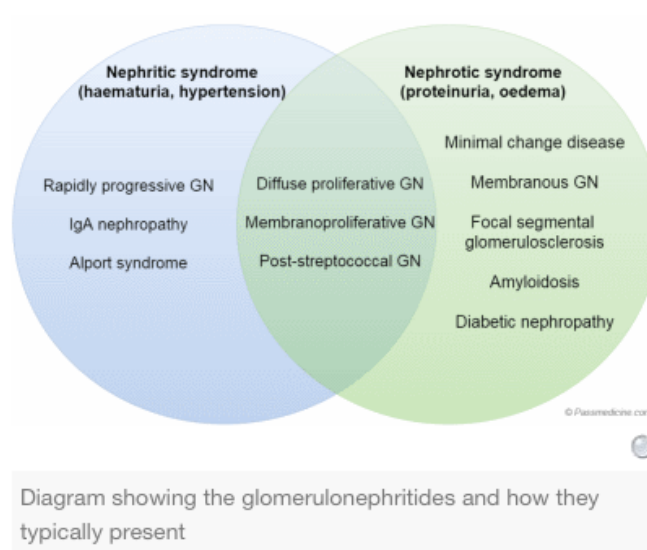


Diagram showing the glomerulonephritides and how they typically present

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Nephrotic syndrome: causes

Primary glomerulonephritis accounts for around 80% of cases

- minimal change glomerulonephritis (causes 80% in children, 30% in adults)
- membranous glomerulonephritis
- focal segmental glomerulosclerosis
- membranoproliferative glomerulonephritis

Systemic disease (about 20%)

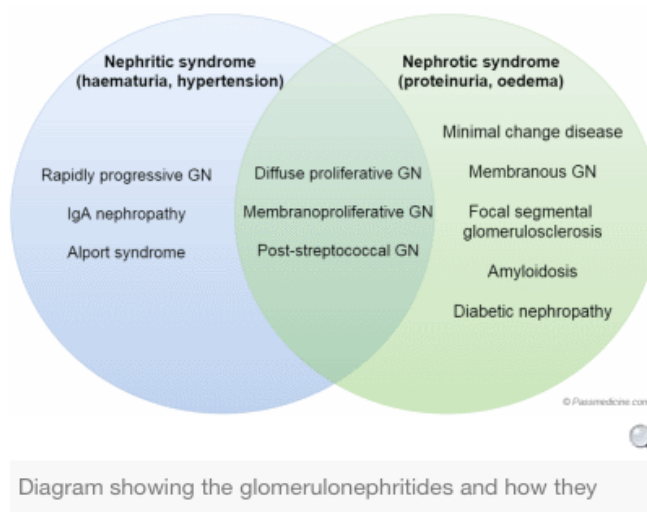
- diabetes mellitus
- systemic lupus erythematosus
- amyloidosis

Drugs

- gold (sodium aurothiomalate), penicillamine

Others

- congenital
- neoplasia: carcinoma, lymphoma, leukaemia, myeloma
- infection: bacterial endocarditis, hepatitis B, malaria



Renal vein thrombosis	46%
Bilateral hydronephrosis	5%
Acute interstitial nephritis	18%
Analgesic nephropathy	8%

Nephrotic syndrome predisposes to thrombotic episodes, possibly due to loss of antithrombin III. These commonly occur in the renal veins and may be bilateral. Common symptoms include loin pain and haematuria.

A greater rise in the ESR would be expected if the renal failure was due to an exacerbation of SLE.

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Nephrotic syndrome: complications

Complications

- increased risk of infection due to urinary immunoglobulin loss
- increased risk of thromboembolism related to loss of antithrombin III and plasminogen in the urine
- hyperlipidaemia
- hypocalcaemia (vitamin D and binding protein lost in urine)
- acute renal failure

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Rapidly progressive glomerulonephritis

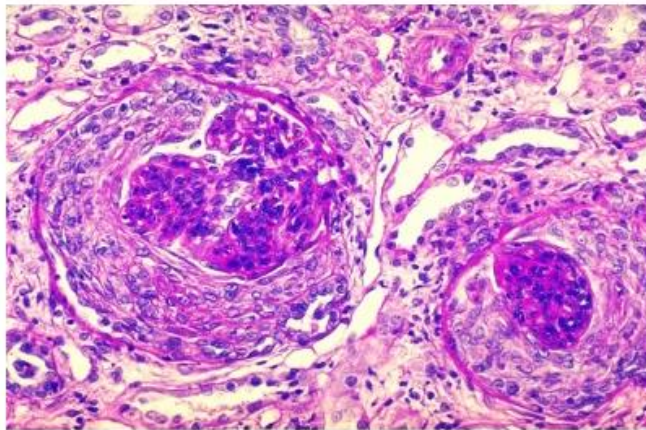
Rapidly progressive glomerulonephritis is a term used to describe a rapid loss of renal function associated with the formation of epithelial crescents in the majority of glomeruli.

Causes

- Goodpasture's syndrome
- Wegener's granulomatosis
- others: SLE, microscopic polyarteritis

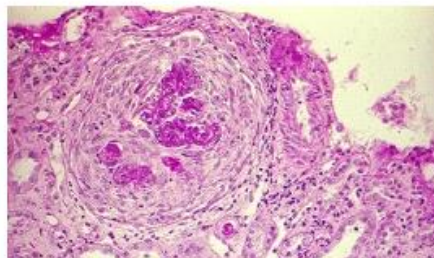
Features

- nephritic syndrome: haematuria with red cell casts, proteinuria, hypertension, oliguria
- features specific to underlying cause (e.g. haemoptysis with Goodpasture's, vasculitic rash or sinusitis with Wegener's)



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Glomeruli are full of crescents.



IgA nephropathy

Basics

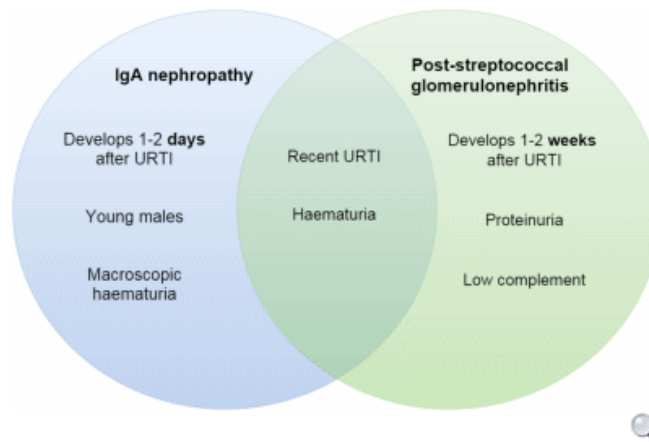
- also called Berger's disease or mesangioproliferative glomerulonephritis
- commonest cause of glomerulonephritis worldwide
- thought to be caused by mesangial deposition of IgA immune complexes
- there is considerable pathological overlap with Henoch-Schonlein purpura (HSP)
- histology: mesangial hypercellularity, positive immunofluorescence for IgA & C3

Presentations

- young male, recurrent episodes of macroscopic haematuria
- typically associated with mucosal infections e.g., URTI
- nephrotic range proteinuria is rare
- renal failure

Differentiating between IgA nephropathy and post-streptococcal glomerulonephritis

- post-streptococcal glomerulonephritis is associated with low complement levels
- main symptom in post-streptococcal glomerulonephritis is proteinuria (although haematuria can occur)
- there is typically an interval between URTI and the onset of renal problems in post-streptococcal glomerulonephritis



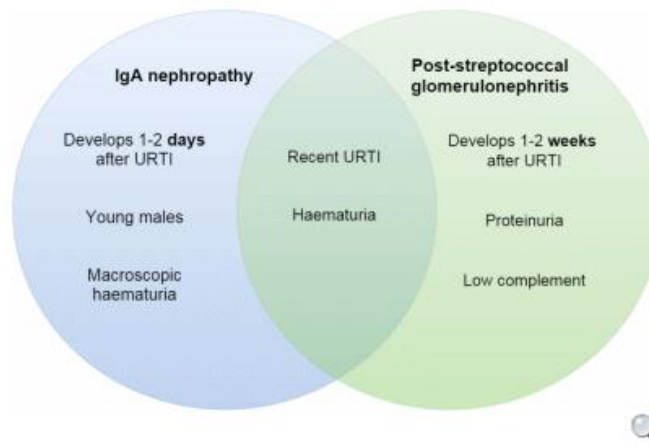
Associated conditions

- alcoholic cirrhosis
- coeliac disease/dermatitis herpetiformis
- Henoch-Schonlein purpura

Management

- steroids/immunosuppressants not be shown to be useful

- post-streptococcal glomerulonephritis is associated with low complement levels
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Associated conditions

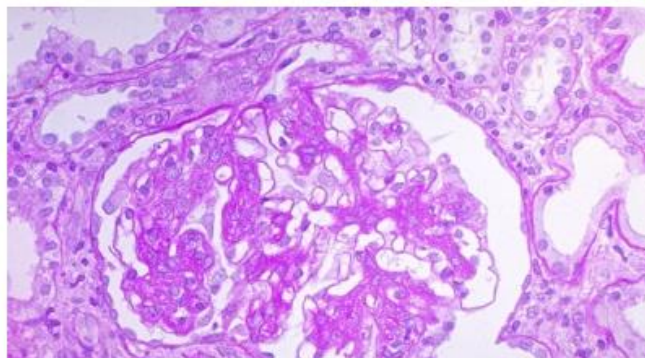
- alcoholic cirrhosis
- coeliac disease/dermatitis herpetiformis
- Henoch-Schonlein purpura

Management

- steroids/immunosuppressants not be shown to be useful

Prognosis

- 25% of patients develop ESRF
- markers of good prognosis: frank haematuria
- markers of poor prognosis: male gender, proteinuria (especially > 2 g/day), hypertension, smoking, hyperlipidaemia, ACE genotype DD



Questions

Post-streptococcal glomerulonephritis



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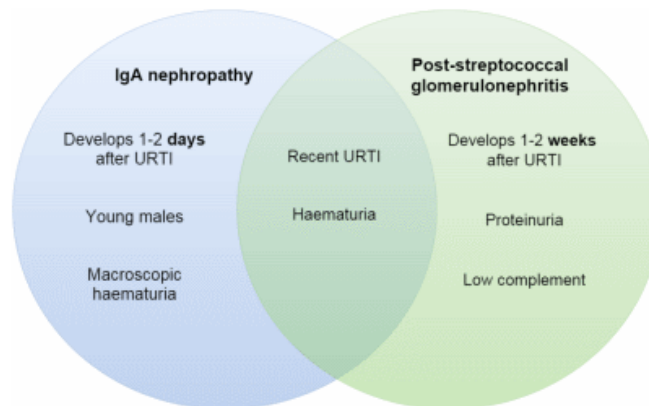
Thicker

appear

Post-streptococcal glomerulonephritis typically occurs 7-14 days following a group A beta-haemolytic *Streptococcus* infection (usually *Streptococcus pyogenes*). It is caused by immune complex (IgG, IgM and C3) deposition in the glomeruli. Young children most commonly affected.

Features

- general: headache, malaise
- haematuria
- proteinuria
- hypertension
- low C3
- raised ASO titre



IgA nephropathy and post-streptococcal glomerulonephritis are often confused as they both can cause renal disease following an URTI

Renal biopsy features

- post-streptococcal glomerulonephritis causes acute, diffuse proliferative glomerulonephritis
- endothelial proliferation with neutrophils
- electron microscopy: subepithelial 'humps' caused by lumpy immune complex deposits
- immunofluorescence: granular or 'starry sky' appearance

Carries a good prognosis



Membranous glomerulonephritis

Membranous glomerulonephritis is the commonest type of glomerulonephritis in adults and is the third most common cause of end-stage renal failure (ESRF). It usually presents with nephrotic syndrome or proteinuria.

Renal biopsy demonstrates:

- electron microscopy: the basement membrane is thickened with subepithelial electron dense deposits. This creates a 'spike and dome' appearance

Causes

- idiopathic
- infections: hepatitis B, malaria, syphilis
- malignancy: lung cancer, lymphoma, leukaemia
- drugs: gold, penicillamine, NSAIDs
- autoimmune diseases: systemic lupus erythematosus (class V disease), thyroiditis, rheumatoid

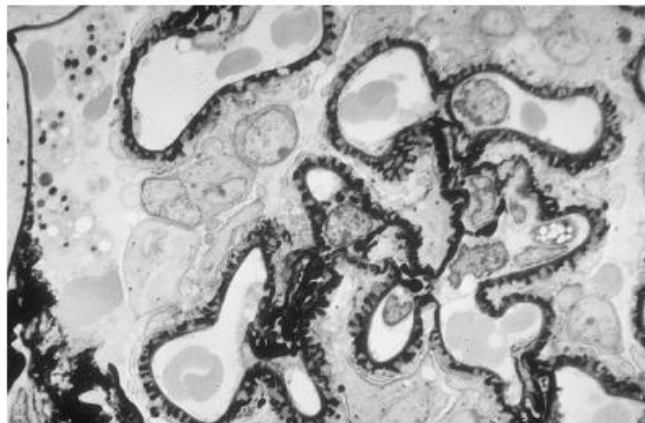
Prognosis - rule of thirds

- one-third: spontaneous remission
- one-third: remain proteinuric
- one-third: develop ESRF

Good prognostic features include female sex, young age at presentation and asymptomatic proteinuria of a modest degree at the time of presentation.

Management

- immunosuppression: corticosteroids alone have not been shown to be effective. A combination of corticosteroid + another agent such as chlorambucil is often used
- blood pressure control: ACE inhibitors have been shown to reduce proteinuria
- consider anticoagulation





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Membranoproliferative glomerulonephritis



Question

Overview

- also known as mesangiocapillary glomerulonephritis
- may present as nephrotic syndrome, haematuria or proteinuria
- poor prognosis

A 30-year-old male presents with haematuria.

Renal p

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Haemo

Type 1

- accounts for 90% of cases
- subendothelial immune deposits of electron dense material resulting in a 'tram-track' appearance
- cause: cryoglobulinaemia, hepatitis C

Type 2 - 'dense deposit disease'

- causes: partial lipodystrophy, factor H deficiency
- reduced serum complement
- C3b nephritic factor (an antibody against C3bBb) found in 70%

Type 3

- causes: hepatitis B and C

Management

- steroids may be effective

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Minimal change disease



Minimal change disease nearly always presents as nephrotic syndrome, accounting for 75% of cases in children and 25% in adults.

The majority of cases are idiopathic, but in around 10-20% a cause is found:

- drugs: NSAIDs, rifampicin
- Hodgkin's lymphoma, thymoma
- infectious mononucleosis

Pathophysiology

- T-cell and cytokine mediated damage to the glomerular basement membrane → polyanion loss
- the resultant reduction of electrostatic charge → increased glomerular permeability to serum albumin

Features

- nephrotic syndrome
- normotension - hypertension is rare
- highly selective proteinuria*
- renal biopsy: electron microscopy shows fusion of podocytes

Management

- majority of cases (80%) are steroid responsive
- cyclophosphamide is the next step for steroid resistant cases

Prognosis is overall good, although relapse is common. Roughly:

- 1/3 have just one episode
- 1/3 have infrequent relapses
- 1/3 have frequent relapses which stop before adulthood

*only intermediate-sized proteins such as albumin and transferrin leak through the glomerulus

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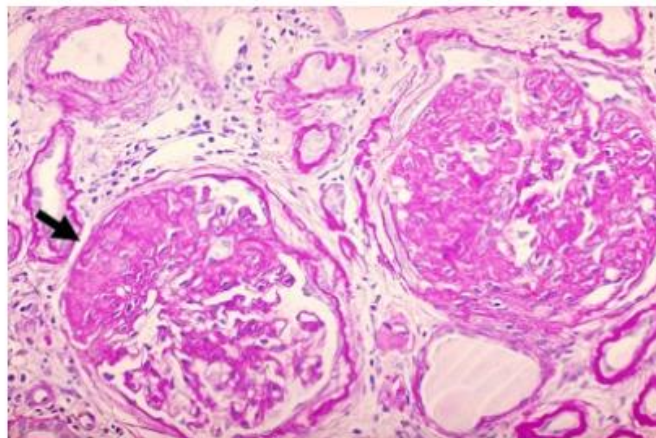
Focal segmental glomerulosclerosis

Focal segmental glomerulosclerosis is cause of nephrotic syndrome and chronic kidney disease. It generally presents in young adults.

Causes

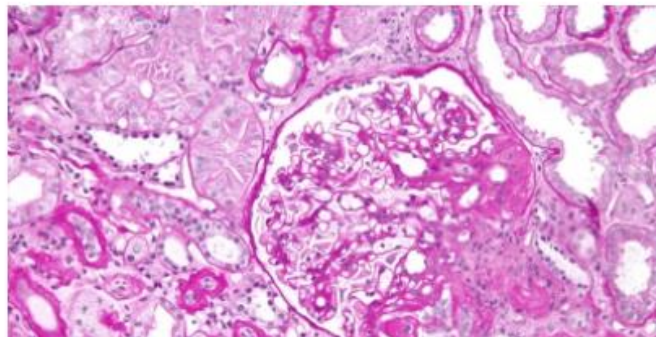
- idiopathic
- secondary to other renal pathology e.g. IgA nephropathy, reflux nephropathy
- HIV
- heroin
- Alport's syndrome
- sickle-cell

Focal segmental glomerulosclerosis is noted for having a high recurrence rate in renal transplants.



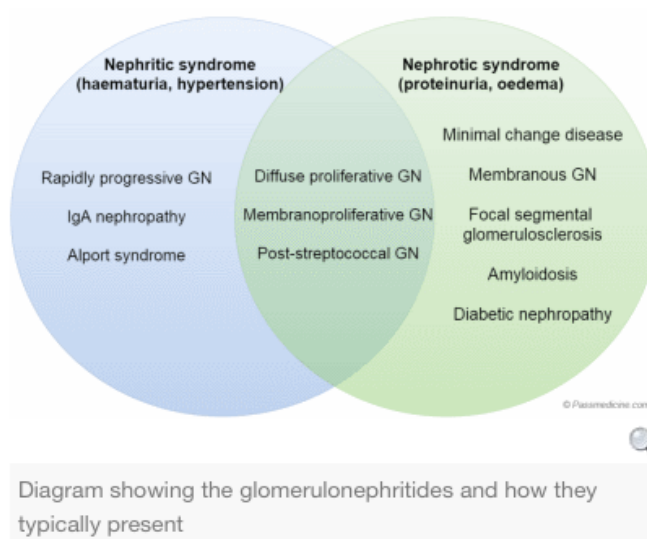
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Sclerosis of the glomerulus is seen next to Bowman's capsule



Glomerulonephritides

Knowing a few key facts is the best way to approach the difficult subject of glomerulonephritis:



Typically presents with nephritic syndrome (haematuria, hypertension)

Rapidly progressive glomerulonephritis - aka crescentic glomerulonephritis

- rapid onset, often presenting as acute kidney injury
- causes include Goodpasture's, ANCA positive vasculitis

IgA nephropathy - aka Berger's disease, mesangioproliferative GN

- typically young adult with haematuria following an URTI

Mixed nephritic/nephrotic presentation

Diffuse proliferative glomerulonephritis

- classical post-streptococcal glomerulonephritis in child
- presents as nephritic syndrome / acute kidney injury
- most common form of renal disease in SLE

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Questions Search Results Normal Values - Reference... Neuropathic pain in adults:...

- typically young adult with haematuria following an URTI

Mixed nephritic/nephrotic presentation

Diffuse proliferative glomerulonephritis

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- presents as nephritic syndrome / acute kidney injury
- most common form of renal disease in SLE

Membranoproliferative glomerulonephritis (mesangiocapillary)

- type 1: cryoglobulinaemia, hepatitis C
- type 2: partial lipodystrophy

Typically presents with nephrotic syndrome (proteinuria, oedema)

Minimal change disease

- typically a child with nephrotic syndrome (accounts for 80%)
- causes: Hodgkin's, NSAIDs
- good response to steroids

Membranous glomerulonephritis

- presentation: proteinuria / nephrotic syndrome / chronic kidney disease
- cause: infections, rheumatoid drugs, malignancy
- 1/3 resolve, 1/3 respond to cytotoxics, 1/3 develop chronic kidney disease

Focal segmental glomerulosclerosis

- may be idiopathic or secondary to HIV, heroin
- presentation: proteinuria / nephrotic syndrome / chronic kidney disease

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time of referral is normal. What is the most likely diagnosis?

Thin basement membrane disease	60%
IgA nephropathy	19%
Renal lithiasis	12%
Acute interstitial nephritis	5%
Post streptococcal glomerulonephritis	4%

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Thin basement membrane disease

An inherited disorder of type IV collagen that causes thinning of the basement membrane. It may affect up to 5% of the population and about 30% patients report a family history of haematuria. Diagnosis is usually based on the history of persistent haematuria, normal kidney function and family history of haematuria without kidney failure. It is generally a benign disorder and biopsy is rarely indicated.

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Systemic lupus erythematosus: renal complications

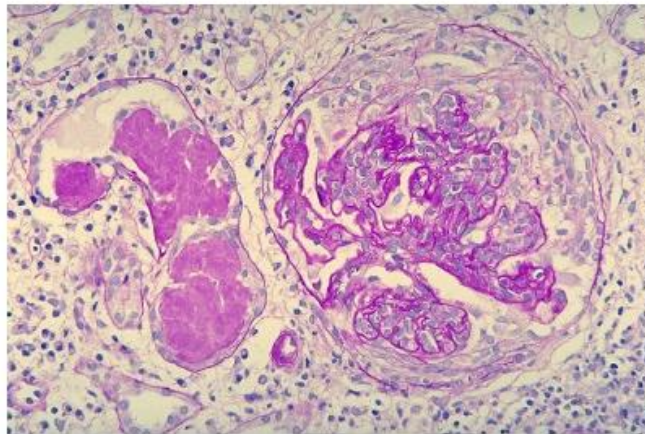
WHO classification

- class I: normal kidney
- class II: mesangial glomerulonephritis
- class III: focal (and segmental) proliferative glomerulonephritis
- class IV: diffuse proliferative glomerulonephritis
- class V: diffuse membranous glomerulonephritis
- class VI: sclerosing glomerulonephritis

Class IV (diffuse proliferative glomerulonephritis) is the most common and severe form.

Renal biopsy characteristically shows the following findings:

- glomeruli shows endothelial and mesangial proliferation, 'wire-loop' appearance
- if severe, the capillary wall may be thickened secondary to immune complex deposition
- electron microscopy shows subendothelial immune complex deposits
- granular appearance on immunofluorescence



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Diffuse proliferative SLE. Proliferation of endothelial and mesangial cells. The thickening of the capillary wall results in a 'wire-loop' appearance. Some crescents are present.

Management

- treat hypertension
- corticosteroids if clinical evidence of disease
- immunosuppressants e.g. azathiopine/cyclophosphamide



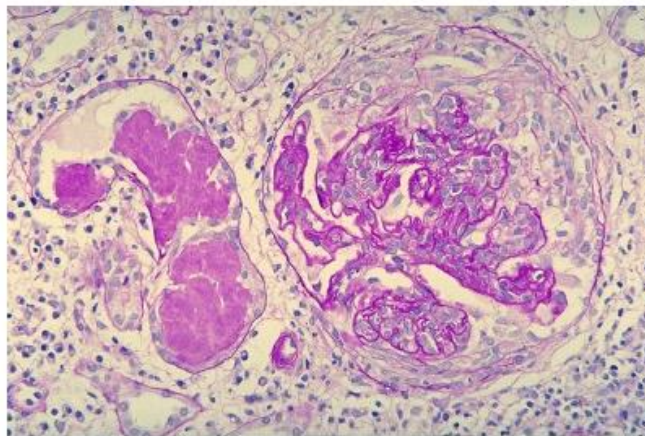
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Diffuse proliferative SLE. Proliferation of endothelial and mesangial cells. The thickening of the capillary wall results in a 'wire-loop' appearance. Some crescents are present.

Management

- treat hypertension
- corticosteroids if clinical evidence of disease
- immunosuppressants e.g. azathiopine/cyclophosphamide



Henoch-Schonlein purpura



Henoch-Schonlein purpura (HSP) is an IgA mediated small vessel vasculitis. There is a degree of overlap with IgA nephropathy (Berger's disease). HSP is usually seen in children following an infection.

Features

- palpable purpuric rash (with localized oedema) over buttocks and extensor surfaces of arms and legs
- abdominal pain
- polyarthrititis
- features of IgA nephropathy may occur e.g. haematuria, renal failure



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Treatment

- analgesia for arthralgia
- treatment of nephropathy is generally supportive. There is inconsistent evidence for the use of steroids and immunosuppressants

Prognosis

- usually excellent, HSP is a self-limiting condition, especially in children without renal involvement
- around 1/3rd of patients have a relapse



Diabetic nephropathy: stages

Diabetic nephropathy may be classified as occurring in five stages*:

Stage 1

- hyperfiltration: increase in GFR
- may be reversible

Stage 2 (silent or latent phase)

- most patients do not develop microalbuminuria for 10 years
- GFR remains elevated

Stage 3 (incipient nephropathy)

- microalbuminuria (albumin excretion of 30 - 300 mg/day, dipstick negative)

Stage 4 (overt nephropathy)

- persistent proteinuria (albumin excretion > 300 mg/day, dipstick positive)
- hypertension is present in most patients
- histology shows diffuse glomerulosclerosis and focal glomerulosclerosis (Kimmelstiel-Wilson nodules)

Stage 5

- end-stage renal disease, GFR typically < 10ml/min
- renal replacement therapy needed

The timeline given here is for type 1 diabetics. Patients with type 2 diabetes mellitus (T2DM) progress through similar stages but in a different timescale - some T2DM patients may progress quickly to the later stages

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Quick comparison: Aetiology (renal disorders)

Generalised disorder of renal tubular transport in the proximal convoluted tubule

- Fanconi syndrome

Mutation on chromosome 16 or 4 affecting renal tubule development

Relevant to my revision >

Less relevant >

Fanconi syndrome

Fanconi syndrome describes a generalised disorder of renal tubular transport in the proximal convoluted tubule resulting in:

- type 2 (proximal) renal tubular acidosis
- polyuria
- aminoaciduria
- glycosuria
- phosphaturia
- osteomalacia

Causes

- cystinosis (most common cause in children)
- Sjogren's syndrome
- multiple myeloma
- nephrotic syndrome
- Wilson's disease

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Goodpasture's syndrome

Goodpasture's syndrome is rare condition associated with both pulmonary haemorrhage and rapidly progressive glomerulonephritis. It is caused by anti-glomerular basement membrane (anti-GBM) antibodies against type IV collagen. Goodpasture's syndrome is more common in men (sex ratio 2:1) and has a bimodal age distribution (peaks in 20-30 and 60-70 age bracket). It is associated with HLA DR2.

Features

- pulmonary haemorrhage
- followed by rapidly progressive glomerulonephritis

Factors which increase likelihood of pulmonary haemorrhage

- smoking
- lower respiratory tract infection
- pulmonary oedema
- inhalation of hydrocarbons
- young males

Investigations

- renal biopsy: linear IgG deposits along basement membrane
- raised transfer factor secondary to pulmonary haemorrhages

Management

- plasma exchange (plasmapheresis)
- steroids
- cyclophosphamide

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Haemolytic uraemic syndrome

Haemolytic uraemic syndrome is generally seen in young children and produces a triad of:

- acute renal failure
- microangiopathic haemolytic anaemia
- thrombocytopenia

Causes

- post-dysentery - classically E coli 0157:H7 ('verotoxigenic', 'enterohaemorrhagic')
- tumours
- pregnancy
- ciclosporin, the Pill
- systemic lupus erythematosus
- HIV

Investigations

- full blood count: anaemia, thrombocytopenia, fragmented blood film
- U&E: acute renal failure
- stool culture

Management

- treatment is supportive e.g. Fluids, blood transfusion and dialysis if required
- there is no role for antibiotics, despite the preceding diarrhoeal illness in many patients
- the indications for plasma exchange in HUS are complicated. As a general rule plasma exchange is reserved for severe cases of HUS not associated with diarrhoea

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Thrombotic thrombocytopenic purpura

Pathogenesis of thrombotic thrombocytopenic purpura (TTP)

- abnormally large and sticky multimers of von Willebrand's factor cause platelets to clump within vessels
- in TTP there is a deficiency of ADAMTS13 (a metalloprotease enzyme) which breakdowns large multimers of von Willebrand's factor
- overlaps with haemolytic uraemic syndrome (HUS)

Features

- rare, typically adult females
- fever
- fluctuating neuro signs (microemboli)
- microangiopathic haemolytic anaemia
- thrombocytopenia
- renal failure

Causes

- post-infection e.g. urinary, gastrointestinal
- pregnancy
- drugs: ciclosporin, oral contraceptive pill, penicillin, clopidogrel, aciclovir
- tumours
- SLE
- HIV

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British Committee for Standards in Haematology

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[2003 TTP/HUS guidelines](#)



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Amyloidosis



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Overview

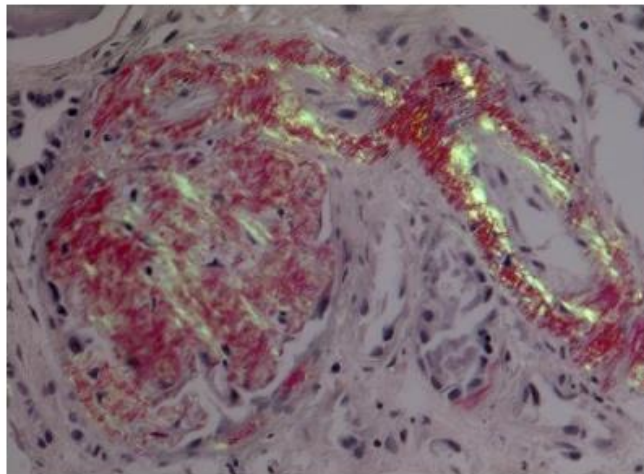
- amyloidosis is a term which describes the extracellular deposition of an insoluble fibrillar protein termed amyloid
- amyloid is derived from many different precursor proteins
- in addition to the fibrillar component, amyloid also contains a non-fibrillary protein called amyloid-P component, derived from the acute phase protein serum amyloid P
- other non-fibrillary components include apolipoprotein E and heparan sulphate proteoglycans
- the accumulation of amyloid fibrils leads to tissue/organ dysfunction

Classification

- systemic or localized
- further characterised by precursor protein (e.g. AL in myeloma - A for Amyloid, L for immunoglobulin Light chain fragments)

Diagnosis

- Congo red staining: apple-green birefringence
- serum amyloid precursor (SAP) scan
- biopsy of rectal tissue



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Renal amyloid with congo red staining - apple-green birefringence



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positive Congo red staining.

Diabetes would be a possibility, however, it is not associated with a positive Congo red staining. AL amyloidosis tends to be associated with an underlying haematological condition as opposed to an inflammatory problem. Again, Waldenstrom macroglobulinaemia and systemic lupus erythematosus would not cause a positive Congo red staining.

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Amyloidosis: types

AL amyloid

- L for immunoglobulin Light chain fragment
- due to myeloma, Waldenstrom's, MGUS
- features include: cardiac and neurological involvement, macroglossia, periorbital ecchymoses

AA amyloid

- A for precursor serum amyloid A protein, an acute phase reactant
- seen in chronic infection/inflammation
- e.g. TB, bronchiectasis, rheumatoid arthritis
- features: renal involvement most common feature

Beta-2 microglobulin amyloidosis

- precursor protein is beta-2 microglobulin, part of the major histocompatibility complex
- associated with patients on renal dialysis

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Normal Values - References Ranges -...

Neuropathic pain in adults: pharmacol...

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Reference ranges

End session

Diabetes insipidus

Diabetes insipidus (DI) is a condition characterised by either a deficiency of antidiuretic hormone, ADH, (cranial DI) or an insensitivity to antidiuretic hormone (nephrogenic DI).

Causes of cranial DI

- idiopathic
- post head injury
- pituitary surgery
- craniopharyngiomas
- histiocytosis X
- DIDMOAD is the association of cranial Diabetes Insipidus, Diabetes Mellitus, Optic Atrophy and Deafness (also known as Wolfram's syndrome)

Causes of nephrogenic DI

- genetic: the more common form affects the vasopression (ADH) receptor, the less common form results from a mutation in the gene that encodes the aquaporin 2 channel
- electrolytes: hypercalcaemia, hypokalaemia
- drugs: demeclocycline, lithium
- tubulo-interstitial disease: obstruction, sickle-cell, pyelonephritis

Features

- polyuria
- polydipsia

Investigation

- high plasma osmolality, low urine osmolality.
- a urine osmolality of >700 mOsm/kg excludes diabetes insipidus.
- water deprivation test

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Nephrogenic diabetes insipidus

(1/1)

Previous radiotherapy	5%
Inflammatory abdominal aortic aneurysm	14%
Methysergide	20%
Sulphonamides	42%

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Retroperitoneal fibrosis

Lower back/flank pain is the most common presenting feature. Fever and lower limb oedema is also seen in some patients.

Associations

- Riedel's thyroiditis
- previous radiotherapy
- sarcoidosis
- inflammatory abdominal aortic aneurysm
- drugs: methysergide

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Search Results Questions

The evidence base is much stronger for volume expansion with normal saline than for N-acetylcysteine.

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Nephrotoxicity due to contrast media

Contrast media nephrotoxicity may be defined as a 25% increase in creatinine occurring within 3 days of the intravascular administration of contrast media.

Risk factors include

- known renal impairment (especially diabetic nephropathy)
- age > 70 years
- dehydration
- cardiac failure
- the use of nephrotoxic drugs such as NSAIDs

Prevention

- the evidence base currently supports the use of intravenous 0.9% sodium chloride at a rate of 1 mL/kg/hour for 12 hours pre- and post- procedure. There is also evidence to support the use of isotonic sodium bicarbonate
- N-acetylcysteine (usually given orally) has been shown to reduce the incidence of contrast-nephropathy in some studies but the evidence base is not as strong as for fluid therapy

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Rhabdomyolysis

Rhabdomyolysis will typically feature in the exam as a patient who has had a fall or prolonged epileptic seizure and is found to have acute renal failure on admission

Features

- acute renal failure with disproportionately raised creatinine
- elevated CK
- myoglobinuria
- hypocalcaemia (myoglobin binds calcium)
- elevated phosphate (released from myocytes)

Causes

- seizure
- collapse/coma (e.g. elderly patients collapses at home, found 8 hours later)
- ecstasy
- crush injury
- McArdle's syndrome
- drugs: statins

Management

- IV fluids to maintain good urine output
- urinary alkalization is sometimes used

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Churg-Strauss would be associated with late-onset asthma, chronic eosinophilic syndrome is a diagnosis of exclusion and is a more long-term event, and arterial thromboembolism would not be associated with eosinophilia. DRESS syndrome would be associated with a drug precipitant which is not mentioned in the question.

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Cholesterol embolisation

Overview

- cholesterol emboli may break off causing renal disease
- seen more commonly in arteriopathies, abdominal aortic aneurysms

Features

- eosinophilia
- purpura
- renal failure
- livedo reticularis

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Flash pulmonary oedema, U&Es worse on ACE inhibitor, asymmetrical kidneys → renal artery stenosis - do MR angiography

There is likely underlying renal artery stenosis revealed by the addition of an ACE inhibitor. Risk factors such as hypertension and hyperlipidaemia which have contributed to the development of his ischaemic heart disease also put him at risk of renal vascular disease

Discuss Improve

Next question >

Renal vascular disease

Renal vascular disease is most commonly due to atherosclerosis (> 95% of patients). It is associated with risk factors such as smoking and hypertension that cause atheroma elsewhere in the body. It may present as hypertension, chronic renal failure or 'flash' pulmonary oedema. In younger patients however fibromuscular dysplasia (FMD) needs to be considered. FMD is more common in young women and characteristically has a 'string of beads' appearance on angiography. Patients respond well to balloon angioplasty

Investigation

- MR angiography is now the investigation of choice
- CT angiography
- conventional renal angiography is less commonly performed used nowadays, but may still have a role when planning surgery

Next question >

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Haematuria

The management of patients with haematuria is often difficult due to the absence of widely followed guidelines. It is sometimes unclear whether patients are best managed in primary care, by urologists or by nephrologists.

The terminology surrounding haematuria is changing. Microscopic or dipstick positive haematuria is increasingly termed non-visible haematuria whilst macroscopic haematuria is termed visible haematuria. Non-visible haematuria is found in around 2.5% of the population.

Causes of transient or spurious non-visible haematuria

- urinary tract infection
- menstruation
- vigorous exercise (this normally settles after around 3 days)
- sexual intercourse

Causes of persistent non-visible haematuria

- cancer (bladder, renal, prostate)
- stones
- benign prostatic hyperplasia
- prostatitis
- urethritis e.g. *Chlamydia*
- renal causes: IgA nephropathy, thin basement membrane disease

Spurious causes - red/orange urine, where blood is not present on dipstick

- foods: beetroot, rhubarb
- drugs: rifampicin, doxorubicin

Management

Current evidence does not support screening for haematuria. The incidence of non-visible haematuria is similar in patients taking aspirin/warfarin to the general population hence these patients should also be investigated.

Testing

.

Management

Current evidence does not support screening for haematuria. The incidence of non-visible haematuria is similar in patients taking aspirin/warfarin to the general population hence these patients should also be investigated.

Testing

- urine dipstick is the test of choice for detecting haematuria
- persistent non-visible haematuria is often defined as blood being present in 2 out of 3 samples tested 2-3 weeks apart
- renal function, albumin:creatinine (ACR) or protein:creatinine ratio (PCR) and blood pressure should also be checked
- urine microscopy may be used but time to analysis significantly affects the number of red blood cells detected

NICE urgent cancer referral guidelines were updated in 2015.

Urgent referral (i.e. within 2 weeks)

Aged ≥ 45 years AND:

- unexplained visible haematuria without urinary tract infection, or
- visible haematuria that persists or recurs after successful treatment of urinary tract infection

Aged ≥ 60 years AND have unexplained nonvisible haematuria and either dysuria or a raised white cell count on a blood test

Non-urgent referral

Aged ≥ 60 years with recurrent or persistent unexplained urinary tract infection

Since the investigation (or not) of non-visible haematuria is such a common dilemma a number of guidelines have been published. They generally agree with NICE guidance, of note:

- patients under the age of 40 years with normal renal function, no proteinuria and who are normotensive do not need to be referred and may be managed in primary care

Next question >

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Proteinuria6%

Focal segmental glomerulosclerosis on renal biopsy13%

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Discuss

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Next question >

HIV: renal involvement

Renal involvement in HIV patients may occur as a consequence of treatment or the virus itself. Protease inhibitors such as indinavir can precipitate intratubular crystal obstruction

HIV-associated nephropathy (HIVAN) accounts for up to 10% of end-stage renal failure cases in the United States. Antiretroviral therapy has been shown to alter the course of the disease.

There are five key features of HIVAN:

- massive proteinuria
- normal or large kidneys
- focal segmental glomerulosclerosis with focal or global capillary collapse on renal biopsy
- elevated urea and creatinine
- normotension

Next question >

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Bendroflumethiazide may help prevent the formation of calcium based renal stones. It may however theoretically increase the risk of urate based stones

  [Discuss](#) [Improve](#)

[Next question >](#)

Renal stones: risk factors

Risk factors

- dehydration
- hypercalciuria, hyperparathyroidism, hypercalcaemia
- cystinuria
- high dietary oxalate
- renal tubular acidosis
- medullary sponge kidney, polycystic kidney disease
- beryllium or cadmium exposure

Risk factors for urate stones

- gout
- ileostomy: loss of bicarbonate and fluid results in acidic urine, causing the precipitation of uric acid

Drug causes

- drugs that promote calcium stones: loop diuretics, steroids, acetazolamide, theophylline
- thiazides can prevent calcium stones (increase distal tubular calcium resorption)

[Next question >](#)

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 Less relevant 

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Renal stones: management

Initial management of renal colic

Medication

- the British Association of Urological Surgeons (BAUS) recommend diclofenac (intramuscular/oral) as the analgesia of choice for renal colic*. This is also supported in the CKS guidelines: '*Administer a parenteral analgesic (such as intramuscular diclofenac) for rapid relief of severe pain*'
- BAUS also endorse the widespread use of alpha-adrenergic blockers to aid ureteric stone passage

Imaging

- BAUS guidelines recommend ultrasound as the initial imaging modality of choice. The sensitivity of ultrasound for stones is around 45% and specificity is around 90%. Complications such as hydronephrosis can also be quickly identified
- following an ultrasound, BAUS recommend a non-contrast CT (NCCT) to confirm the diagnosis. 99% of stones are identifiable on NCCT. Some GPs now have direct access to NCCT

Stones < 5 mm will usually pass spontaneously. Lithotripsy and nephrolithotomy may be for severe cases.

Management of renal stones

Most renal stones measuring less than 5mm in maximum diameter will typically pass within 4 weeks of symptom onset. More intensive and urgent treatment is indicated in the presence of ureteric obstruction, renal developmental abnormality such as horseshoe kidney and previous renal transplant. Ureteric obstruction due to stones together with infection is a surgical emergency and the system must be decompressed. Options include nephrostomy tube placement, insertion of ureteric catheters and ureteric stent placement.

In the non emergency setting the preferred options for treatment of stone disease include extra corporeal shock wave lithotripsy, percutaneous nephrolithotomy, ureteroscopy, open surgery remains an option for selected cases. However, minimally invasive options are the most popular first line treatment.

Shock wave lithotripsy

- A shock wave is generated external to the patient, internally cavitation bubbles and mechanical stress lead to stone fragmentation. The passage of shock waves can result in the development of solid organ injury. Fragmentation of larger stones may result in the development of ureteric obstruction. The procedure is uncomfortable for patients and analgesia is required during the procedure and afterwards.

Ureteroscopy

- A ureteroscope is passed retrograde through the ureter and into the renal pelvis. It is indicated in individuals (e.g. pregnant females) where lithotripsy is contraindicated and in complex stone disease. In most cases a stent is left in situ for 4 weeks after the procedure.

Percutaneous nephrolithotomy

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Ureteroscopy

- A ureteroscope is passed retrograde through the ureter and into the renal pelvis. It is indicated in individuals (e.g. pregnant females) where lithotripsy is contraindicated and in complex stone disease. In most cases a stent is left in situ for 4 weeks after the procedure.

Percutaneous nephrolithotomy

- In this procedure access is gained to the renal collecting system. Once access is achieved, intra corporeal lithotripsy or stone fragmentation is performed and stone fragments removed.

Therapeutic selection

Disease	Option
Stone burden of less than 2cm in aggregate	Lithotripsy
Stone burden of less than 2cm in pregnant females	Ureteroscopy
Complex renal calculi and staghorn calculi	Percutaneous nephrolithotomy
Ureteric calculi less than 5mm	Manage expectantly

Prevention of renal stones

Calcium stones may be due to hypercalciuria, which is found in up to 5-10% of the general population.

- high fluid intake
- low animal protein, low salt diet (a low calcium diet has not been shown to be superior to a normocalcaemic diet)
- thiazides diuretics (increase distal tubular calcium resorption)

Oxalate stones

- cholestyramine reduces urinary oxalate secretion
- pyridoxine reduces urinary oxalate secretion

Uric acid stones

- allopurinol
- urinary alkalinization e.g. oral bicarbonate

* it should be remembered that diclofenac, in general, is now less commonly used following the MHRA warnings about cardiovascular risk.

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Questions

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Renal stones: imaging

The table below summarises the appearance of different types of renal stone on x-ray

Type	Frequency	Radiograph appearance
Calcium oxalate	40%	Opaque
Mixed calcium oxalate/phosphate stones	25%	Opaque
Triple phosphate stones*	10%	Opaque
Calcium phosphate	10%	Opaque
Urate stones	5-10%	Radio-lucent
Cystine stones	1%	Semi-opaque, 'ground-glass' appearance
Xanthine stones	<1%	Radio-lucent

*stag-horn calculi involve the renal pelvis and extend into at least 2 calyces. They develop in alkaline urine and are composed of struvite (ammonium magnesium phosphate, triple phosphate). Ureaplasma urealyticum and Proteus infections predispose to their formation

Next question >

B I

Hypokalaemia and hypertension

For exams it is useful to be able to classify the causes of hypokalaemia in to those associated with hypertension, and those which are not

Hypokalaemia with hypertension

- Cushing's syndrome
- Conn's syndrome (primary hyperaldosteronism)
- Liddle's syndrome
- 11-beta hydroxylase deficiency*

Carbenoxolone, an anti-ulcer drug, and liquorice excess can potentially cause hypokalaemia associated with hypertension

Hypokalaemia without hypertension

- diuretics
- GI loss (e.g. Diarrhoea, vomiting)
- renal tubular acidosis (type 1 and 2**)
- Bartter's syndrome
- Gitelman syndrome

*21-hydroxylase deficiency, which accounts for 90% of congenital adrenal hyperplasia cases, is not associated with hypertension

**type 4 renal tubular acidosis is associated with hyperkalaemia

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Oral calcium resonium 5%

Calcium gluconate would stabilise the myocardium but would not reduce the serum potassium level.

  Discuss (3) Improve

Next question >

Hyperkalaemia: management

Untreated hyperkalaemia may cause life-threatening arrhythmias. Precipitating factors should be addressed (e.g. acute renal failure) and aggravating drugs stopped (e.g. ACE inhibitors). Management may be categorised by the aims of treatment

Stabilisation of the cardiac membrane

- intravenous calcium gluconate

Short-term shift in potassium from extracellular to intracellular fluid compartment

- combined insulin/dextrose infusion
- nebulised salbutamol

Removal of potassium from the body

- calcium resonium (orally or enema)
- loop diuretics
- dialysis

Next question >

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Spironolactone

Spironolactone is an aldosterone antagonist which acts in the cortical collecting duct.

Indications

- ascites: patients with cirrhosis develop a secondary hyperaldosteronism. Relatively large doses such as 100 or 200mg are often used
- hypertension: used in some patients as a NICE 'step 4' treatment
- heart failure (see RALES study below)
- nephrotic syndrome
- Conn's syndrome

Adverse effects

- hyperkalaemia
- gynaecomastia

RALES

- NYHA III + IV, patients already taking ACE inhibitor
- low dose spironolactone reduces all cause mortality

Save my notes

Close

A 5-year-old boy develops a palpable, erythematous rash on the extensor surfaces of the arms and legs associated with abdominal pain. He is later found to have haematuria and mild renal failure - Henoch-Schonlein purpura

Notes (1/1)

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Less relevant

Metabolic acidosis

Metabolic acidosis is commonly classified according to the anion gap. This can be calculated by: $(\text{Na}^+ + \text{K}^+) - (\text{Cl}^- + \text{HCO}_3^-)$. If a question supplies the chloride level then this is often a clue that the anion gap should be calculated. The normal range = 10-18 mmol/L

Normal anion gap (= hyperchloraemic metabolic acidosis)

- gastrointestinal bicarbonate loss: diarrhoea, ureterosigmoidostomy, fistula
- renal tubular acidosis
- drugs: e.g. acetazolamide
- ammonium chloride injection
- Addison's disease

Raised anion gap

- lactate: shock, hypoxia
- ketones: diabetic ketoacidosis, alcohol
- urate: renal failure
- acid poisoning: salicylates, methanol

Metabolic acidosis secondary to high lactate levels may be subdivided into two types:

- lactic acidosis type A: shock, hypoxia, burns
- lactic acidosis type B: metformin

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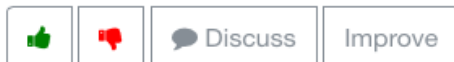
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The anion gap may be calculated by using (sodium + potassium) - (bicarbonate + chloride)

$$= (142 + 4.0) - (104 + 19) = 23 \text{ mmol/L}$$



Next question >

Anion gap

The anion gap is calculated by:

(sodium + potassium) - (bicarbonate + chloride)

A normal anion gap is 8-14 mmol/L

It is useful to consider in patients with a metabolic acidosis:

Causes of a normal anion gap or hyperchloraemic metabolic acidosis

- gastrointestinal bicarbonate loss: diarrhoea, ureterosigmoidostomy, fistula
- renal tubular acidosis
- drugs: e.g. acetazolamide
- ammonium chloride injection
- Addison's disease

Causes of a raised anion gap metabolic acidosis

- lactate: shock, hypoxia
- ketones: diabetic ketoacidosis, alcohol
- urate: renal failure
- acid poisoning: salicylates, methanol

Next question >

Lower urinary tract symptoms in men

Lower urinary tract symptoms (LUTS) in men are very common and are present in the majority of men aged > 50 years. They are most commonly secondary to benign prostatic hyperplasia but other causes should be considered including prostate cancer.

It is useful to classify the symptoms into 3 broad groups.

Voiding	Storage	Post-micturition
Hesitancy Poor or intermittent stream Straining Incomplete emptying Terminal dribbling	Urgency Frequency Nocturia Urinary incontinence	Post-micturition dribbling Sensation of incomplete emptying

Examination

- urinalysis: exclude infection, check for haematuria
- digital rectal examination: size and consistency of prostate
- a PSA test may be indicated, but the patient should be properly counselled first

It is useful to get the patient to complete the following to guide management:

- urinary frequency-volume chart: distinguish between urinary frequency, polyuria, nocturia, and nocturnal polyuria.
- International Prostate Symptom Score (IPSS): assess the impact on the patient's life. This classifies the symptoms as mild, moderate or severe

Management

Predominately voiding symptoms

- conservative measures include: pelvic floor muscle training, bladder training, prudent fluid intake and containment products
- if 'moderate' or 'severe' symptoms offer an alpha-blocker
- if the prostate is enlarged and the patient is 'considered at high risk of progression' then a

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Questions

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Neuropathic pain in adults: pharmacol...

- international Prostate Symptom Score (IPSS): assess the impact on the patient's life. This classifies the symptoms as mild, moderate or severe

Management

Predominately voiding symptoms

- conservative measures include: pelvic floor muscle training, bladder training, prudent fluid intake and containment products
- if 'moderate' or 'severe' symptoms offer an alpha-blocker
- if the prostate is enlarged and the patient is 'considered at high risk of progression' then a 5-alpha reductase inhibitor should be offered
- if the patient has an enlarged prostate and 'moderate' or 'severe' symptoms offer both an alpha-blocker and 5-alpha reductase inhibitor
- if there are mixed symptoms of voiding and storage not responding to an alpha blocker then a antimuscarinic (anticholinergic) drug may be added

Predominately overactive bladder

- conservative measures include moderating fluid intake
- bladder retraining should be offered
- antimuscarinic drugs should be offered if symptoms persist. NICE recommend oxybutynin (immediate release), tolterodine (immediate release), or darifenacin (once daily preparation)
- mirabegron may be considered if first-line drugs fail

Nocturia

- advise about moderating fluid intake at night
- furosemide 40mg in late afternoon may be considered
- desmopressin may also be helpful

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Relevant to my revision

Less relevant

ARPKD

Autosomal recessive polycystic kidney disease (ARPKD) is much less common than autosomal dominant disease (ADPKD). It is due to a defect in a gene located on chromosome 6 which encodes fibrocystin, a protein important for normal renal tubule development.

Diagnosis may be made on prenatal ultrasound or in early infancy with abdominal masses and renal failure. Newborns may also have features consistent with Potter's syndrome secondary to oligohydramnios. End-stage renal failure develops in childhood. Patients also typically have liver involvement, for example portal and interlobular fibrosis.

Renal biopsy typically shows multiple cylindrical lesions at right angles to the cortical surface.

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ADPKD

Autosomal dominant polycystic kidney disease (ADPKD) is the most common inherited cause of kidney disease, affecting 1 in 1,000 Caucasians. Two disease loci have been identified, PKD1 and PKD2, which code for polycystin-1 and polycystin-2 respectively

ADPKD type 1	ADPKD type 2
85% of cases	15% of cases
Chromosome 16	Chromosome 4
Presents with renal failure earlier	

The screening investigation for relatives is abdominal ultrasound:

Ultrasound diagnostic criteria (in patients with positive family history)

- two cysts, unilateral or bilateral, if aged < 30 years
- two cysts in both kidneys if aged 30-59 years
- four cysts in both kidneys if aged > 60 years

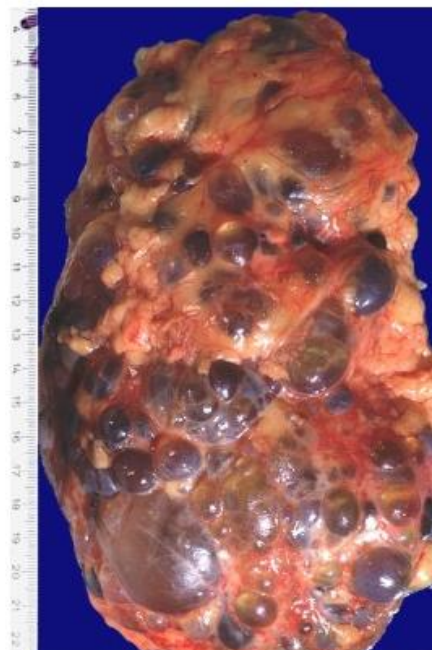


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Questions Chronic kidney disease in a... Normal Values - Reference... Neuropathic pain in adults:...

Next question >

ADPKD: features

Features

- hypertension
- recurrent UTIs
- abdominal pain
- renal stones
- haematuria
- chronic kidney disease

Extra-renal manifestations

- liver cysts (70%)
- berry aneurysms (8%)
- cardiovascular system: mitral valve prolapse, mitral/tricuspid incompetence, aortic root dilation, aortic dissection
- cysts in other organs: pancreas, spleen; very rarely: thyroid, oesophagus, ovary





Renal papillary necrosis

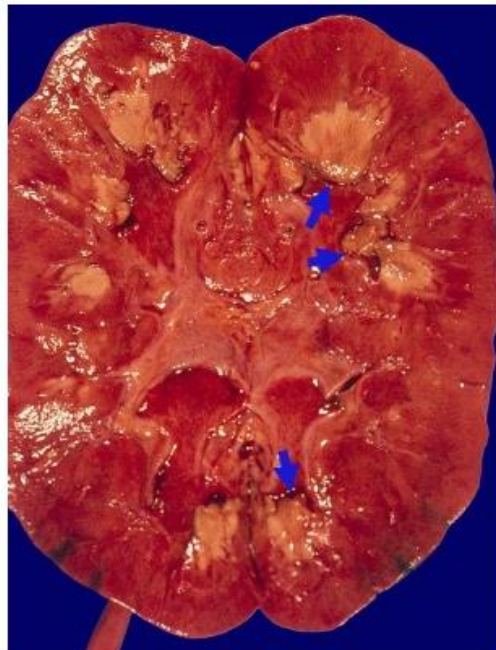
Renal papillary necrosis describes the coagulative necrosis of the renal papillae due to a variety of causes.

Features

- visible haematuria
- loin pain
- proteinuria

Causes

- severe acute pyelonephritis
- diabetic nephropathy
- obstructive nephropathy
- analgesic nephropathy: phenacetin was the classic cause but this has now been withdrawn
- sickle cell anaemia



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Renal papillary necrosis secondary to acute pyelonephritis. Note the pale areas highlighted by the blue arrows

Next question >

Acute kidney injury: acute tubular necrosis vs. prerenal uraemia

Prerenal uraemia - kidneys hold on to sodium to preserve volume

	Pre-renal uraemia	Acute tubular necrosis
Urine sodium	< 20 mmol/L	> 30 mmol/L
Fractional sodium excretion*	< 1%	> 1%
Fractional urea excretion**	< 35%	>35%
Urine:plasma osmolality	> 1.5	< 1.1
Urine:plasma urea	> 10:1	< 8:1
Specific gravity	> 1020	< 1010
Urine	'bland' sediment	brown granular casts
Response to fluid challenge	Yes	No

*fractional sodium excretion = (urine sodium/plasma sodium) / (urine creatinine/plasma creatinine) x 100

**fractional urea excretion = (urine urea /blood urea) / (urine creatinine/plasma creatinine) x 100

Next question >

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Tuberculosis 13%

Chronic analgesia use 13%

Syphilis 44%

Sickle cell disease 13%



Next question >

Papillary necrosis

Causes

- chronic analgesia use
- sickle cell disease
- TB
- acute pyelonephritis
- diabetes mellitus

Features

- fever, loin pain, haematuria
- IVU - papillary necrosis with renal scarring - 'cup & spill'

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Fluid therapy

The prescription of intravenous fluids is one of the most common tasks that junior doctors need to do.

In the 2013 guidelines NICE recommend the following requirements for maintenance fluids:

- 25-30 ml/kg/day of water and
- approximately 1 mmol/kg/day of potassium, sodium and chloride and
- approximately 50-100 g/day of glucose to limit starvation ketosis

So, for a 80kg patient, for a 24 hour period, this would translate to:

- 2 litres of water
- 80mmol potassium

For the first 24 hours NICE recommend the following::

When prescribing for routine maintenance alone, consider using 25-30 ml/kg/day sodium chloride 0.18% in 4% glucose with 27 mmol/l potassium on day 1 (there are other regimens to achieve this).

The amount of fluid patients require obviously varies according to their recent and past medical history. For example a patient who is post-op and is having significant losses from drains will require more fluid whereas a patient with heart failure should be given less fluid to avoid precipitating pulmonary oedema.

The table below shows the electrolyte concentrations (in millimoles/litre) of plasma and the most commonly used fluids:

	Na ⁺	Cl ⁻	K ⁺	HCO ₃ ⁻	Glucose
Plasma	135-145	98-105	3.5-5	22-28	-
0.9% saline	154	154	-	-	-
5% dextrose	-	-	-	-	50

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80mmol potassium
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Questions

When prescribing for routine maintenance alone, consider using 25-30 ml/kg/day sodium chloride 0.18% in 4% glucose with 27 mmol/l potassium on day 1 (there are other regimens to achieve this).

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The table below shows the electrolyte concentrations (in millimoles/litre) of plasma and the most commonly used fluids:

	Na ⁺	Cl ⁻	K ⁺	HCO ₃ ⁻	Glucose
Plasma	135-145	98-105	3.5-5	22-28	-
0.9% saline	154	154	-	-	-
5% glucose	-	-	-	-	50g
0.18% saline with 4% glucose	30	30	-	-	40g
Hartmann's solution	131	111	5	29	-

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Questions

Escherichia coli

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Peritoneal dialysis

Peritoneal dialysis (PD) is a form of renal replacement therapy. It is sometimes used as a stop-gap to haemodialysis or for younger patients who do not want to have to visit hospital three times a week.

The majority of patients do Continuous Ambulatory Peritoneal Dialysis (CAPD), which involves four 2-litre exchanges/day.

Complications

- peritonitis: coagulase-negative staphylococci such as *Staphylococcus epidermidis* is the most common cause. *Staphylococcus aureus* is another common cause
- sclerosing peritonitis

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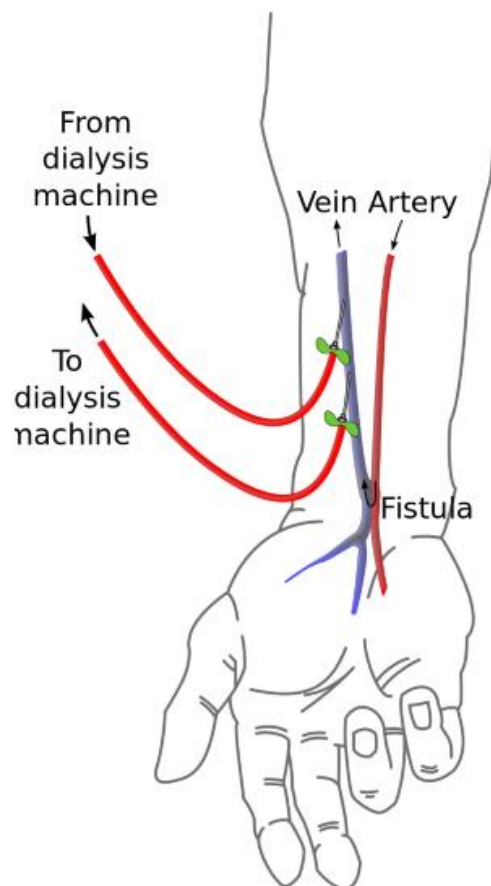
Arteriovenous fistulas

Arteriovenous fistulas are direct connections between arteries and veins. They may occur pathologically but are generally formed surgically to allow access for haemodialysis.

They are now regarded as the preferred method of access for haemodialysis due to the lower rates of complications.

Potential complications include:

- infection
- thrombosis
- stenosis
- steal syndrome



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Questions

Normal Values - References Ranges -...

Neuropathic pain in adults: pharmacol...

Add a beta-blocker 12%

Add spironolactone 12%

Stop ramipril 12%

As the eGFR is 29 ml/min switching bendroflumethiazide to furosemide would be the next step in controlling his blood pressure. Please see the guidelines in the external links section

Discuss (1)
 Improve

Next question >

Chronic kidney disease: hypertension

The majority of patients with chronic kidney disease (CKD) will require more than two drugs to treat hypertension. ACE inhibitors are first line and are particularly helpful in proteinuric renal disease (e.g. diabetic nephropathy). As these drugs tend to reduce filtration pressure a small fall in glomerular filtration pressure (GFR) and rise in creatinine can be expected. NICE suggest that a decrease in eGFR of up to 25% or a rise in creatinine of up to 30% is acceptable, although any rise should prompt careful monitoring and exclusion of other causes (e.g. NSAIDs). A rise greater than this may indicate underlying renovascular disease.

Furosemide is useful as a anti-hypertensive in patients with CKD, particularly when the GFR falls to below 45 ml/min*. It has the added benefit of lowering serum potassium. High doses are usually required. If the patient becomes at risk of dehydration (e.g. Gastroenteritis) then consideration should be given to temporarily stopping the drug

*the NKF K/DOQI guidelines suggest a lower cut-off of less than 30 ml/min

Next question >

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Chronic kidney disease: proteinuria

Proteinuria is an important marker of chronic kidney disease, especially for diabetic nephropathy. NICE recommend using the albumin:creatinine ratio (ACR) in preference to the protein:creatinine ratio (PCR) when identifying patients with proteinuria as it has greater sensitivity. For quantification and monitoring of proteinuria, PCR can be used as an alternative, although ACR is recommended in diabetics. Urine reagent strips are not recommended unless they express the result as an ACR

Approximate equivalent values

ACR (mg/mmol)	PCR (mg/mmol)	Urinary protein excretion (g/24 h)
30	50	0.5
70	100	1

Collecting an ACR sample

- by collecting a 'spot' sample it avoids the need to collect urine over a 24 hour period in order to detect or quantify proteinuria
- should be a first-pass morning urine specimen
- if the initial ACR is between 3 mg/mmol and 70 mg/mmol, this should be confirmed by a subsequent early morning sample. If the initial ACR is 70 mg/mmol or more, a repeat sample need not be tested.

Interpreting the ACR results

- the NICE guidelines state 'regard a confirmed ACR of 3 mg/mmol or more as clinically important proteinuria'

NICE recommendations for referral to a nephrologist:

- a urinary albumin:creatinine ratio (ACR) of 70 mg/mmol or more, unless known to be caused by diabetes and already appropriately treated
- a urinary ACR of 30 mg/mmol or more, together with persistent haematuria (two out of three dipstick tests show 1+ or more of blood) after exclusion of a urinary tract infection
- consider referral to a nephrologist for people with an ACR between 3-69 mg/mmol who

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Questions

Chronic kidney disease in a... Normal Values - Reference... Neuropathic pain in adults:...

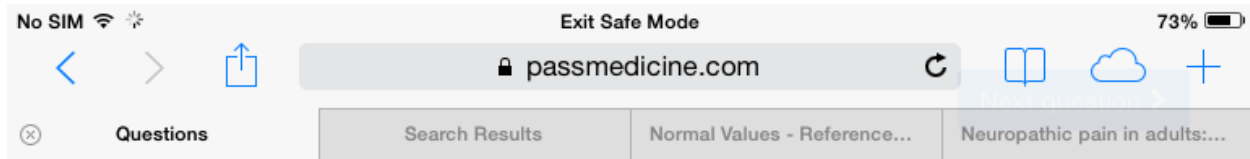
NICE recommendations for referral to a nephrologist:

- a urinary albumin:creatinine ratio (ACR) of 70 mg/mmol or more, unless known to be caused by diabetes and already appropriately treated
- a urinary ACR of 30 mg/mmol or more, together with persistent haematuria (two out of three dipstick tests show 1+ or more of blood) after exclusion of a urinary tract infection
- consider referral to a nephrologist for people with an ACR between 3-29 mg/mmol who have persistent haematuria and other risk factors such as a declining eGFR, or cardiovascular disease

Frequency of monitoring eGFR (number of times per year by eGFR and ACR categories) for people with or at risk of CKD

eGFR categories (mL/min/1.73 m ²)	ACR categories (mg/mmol)		
	A1 (< 3) Normal to mildly increased	A2 (3-30) Moderately increased	A3 (> 30) Severely increased
G1 ≥90 Normal and high	≤ 1	1	≥ 1
G2 60-89 Mild reduction related to normal range for a young adult	≤ 1	1	≥ 1
G3a 45-59 Mild to moderate reduction	1	1	2
G3b 30-44 Moderate to severe reduction	≤ 2	2	≥ 2
G4 15-29 Severe reduction	2	2	3
G5 <15 Kidney failure	4	≥ 4	≥ 4

Next question >



Renal transplant: HLA typing and graft failure

The human leucocyte antigen (HLA) system is the name given to the major histocompatibility complex (MHC) in humans. It is coded for on chromosome 6.

Some basic points on the HLA system

- class 1 antigens include A, B and C. Class 2 antigens include DP, DQ and DR
- when HLA matching for a renal transplant the relative importance of the HLA antigens are as follows DR > B > A

Graft survival

- 1 year = 90%, 10 years = 60% for cadaveric transplants
- 1 year = 95%, 10 years = 70% for living-donor transplants

Post-op problems

- ATN of graft
- vascular thrombosis
- urine leakage
- UTI

Hyperacute acute rejection (minutes to hours)

- due to pre-existent antibodies against donor HLA type 1 antigens (a type II hypersensitivity reaction)
- rarely seen due to HLA matching

Acute graft failure (< 6 months)

- usually due to mismatched HLA. Cell-mediated (cytotoxic T cells)
- other causes include cytomegalovirus infection
- may be reversible with steroids and immunosuppressants

Causes of chronic graft failure (> 6 months)

- both antibody and cell mediated mechanisms cause fibrosis to the transplanted kidney (chronic allograft nephropathy)
- recurrence of original renal disease (MCGN > IgA > FSGS)

Chronic kidney disease: eGFR and classification

Serum creatinine may not provide an accurate estimate of renal function due to differences in muscle. For this reason formulas were developed to help estimate the glomerular filtration rate (estimated GFR or eGFR). The most commonly used formula is the Modification of Diet in Renal Disease (MDRD) equation, which uses the following variables:

- serum creatinine
- age
- gender
- ethnicity

Factors which may affect the result

- pregnancy
- muscle mass (e.g. amputees, body-builders)
- eating red meat 12 hours prior to the sample being taken

CKD may be classified according to GFR:

CKD stage	GFR range
1	Greater than 90 ml/min, with some sign of kidney damage on other tests (if all the kidney tests* are normal, there is no CKD)
2	60-90 ml/min with some sign of kidney damage (if kidney tests* are normal, there is no CKD)
3a	45-59 ml/min, a moderate reduction in kidney function
3b	30-44 ml/min, a moderate reduction in kidney function
4	15-29 ml/min, a severe reduction in kidney function
5	Less than 15 ml/min, established kidney failure - dialysis or a kidney transplant may be needed

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4	15-29 ml/min, a severe reduction in kidney function
5	Less than 15 ml/min, established kidney failure - dialysis or a kidney transplant may be needed

*i.e. normal U&Es and no proteinuria

Next question >

Chronic kidney disease: anaemia

Patients with chronic kidney disease (CKD) may develop anaemia due to a variety of factors, the most significant of which is reduced erythropoietin levels. This is usually a normochromic normocytic anaemia and becomes apparent when the GFR is less than 35 ml/min (other causes of anaemia should be considered if the GFR is > 60 ml/min). Anaemia in CKD predisposes to the development of left ventricular hypertrophy - associated with a three fold increase in mortality in renal patients

Causes of anaemia in renal failure

- reduced erythropoietin levels - the most significant factor
- reduced erythropoiesis due to toxic effects of uraemia on bone marrow
- reduced absorption of iron
- anorexia/nausea due to uraemia
- reduced red cell survival (especially in haemodialysis)
- blood loss due to capillary fragility and poor platelet function
- stress ulceration leading to chronic blood loss

Management

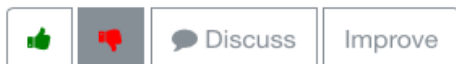
- the 2011 NICE guidelines suggest a target haemoglobin of 10 - 12 g/dl
- determination and optimisation of iron status should be carried out prior to the administration of erythropoiesis-stimulating agents (ESA). Many patients, especially those on haemodialysis, will require IV iron
- ESAs such as erythropoietin and darbepoetin should be used in those 'who are likely to benefit in terms of quality of life and physical function'

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Erythropoietin

Erythropoietin is a haematopoietic growth factor that stimulates the production of erythrocytes. The main uses of erythropoietin are to treat the anaemia associated with chronic kidney disease and that associated with cytotoxic therapy.

Side-effects of erythropoietin

- accelerated hypertension potentially leading to encephalopathy and seizures (blood pressure increases in 25% of patients)
- bone aches
- flu-like symptoms
- skin rashes, urticaria
- pure red cell aplasia* (due to antibodies against erythropoietin)
- raised PCV increases risk of thrombosis (e.g. Fistula)
- iron deficiency 2nd to increased erythropoiesis

There are a number of reasons why patients may fail to respond to erythropoietin therapy:

- iron deficiency
- inadequate dose
- concurrent infection/inflammation
- hyperparathyroid bone disease
- aluminium toxicity

*the risk is greatly reduced with darbepoetin

Next question >



Chronic kidney disease: bone disease

Basic problems in chronic kidney disease

- low vitamin D (1-alpha hydroxylation normally occurs in the kidneys)
- high phosphate
- low calcium: due to lack of vitamin D, high phosphate
- secondary hyperparathyroidism: due to low calcium, high phosphate and low vitamin D

Several clinical manifestations may result:

Osteitis fibrosa cystica

- aka hyperparathyroid bone disease

Adynamic

- reduction in cellular activity (both osteoblasts and osteoclasts) in bone
- may be due to over treatment with vitamin D

Osteomalacia

- due to low vitamin D

Osteosclerosis

Osteoporosis





Renal cell cancer

Renal cell cancer is also known as hypernephroma and accounts for 85% of primary renal neoplasms. It arises from proximal renal tubular epithelium

Associations*

- more common in middle-aged men
- smoking
- von Hippel-Lindau syndrome
- tuberous sclerosis

Features

- classical triad: haematuria, loin pain, abdominal mass
- pyrexia of unknown origin
- left varicocele (due to occlusion of left testicular vein)
- endocrine effects: may secrete erythropoietin (polycythaemia), parathyroid hormone (hypercalcaemia), renin, ACTH
- 25% have metastases at presentation

Management

- for confined disease a partial or total nephrectomy depending on the tumour size
- alpha-interferon and interleukin-2 have been used to reduce tumour size and also treat patients with metastases
- receptor tyrosine kinase inhibitors (e.g. sorafenib, sunitinib) have been shown to have superior efficacy compared to interferon-alpha





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Coronal CT scan of a middle-aged woman with renal cell cancer. Note the heterogeneously enhancing mass at the upper pole of the right kidney

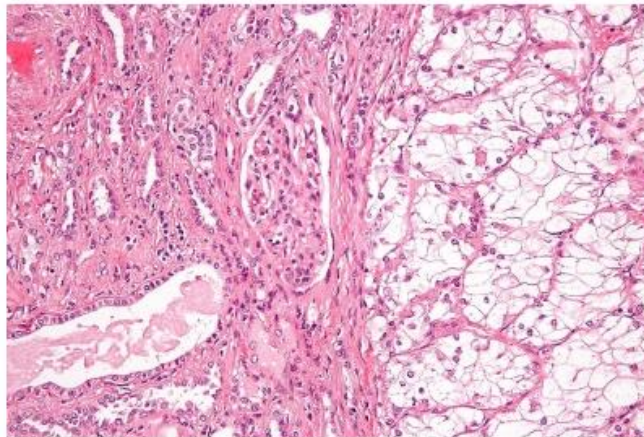
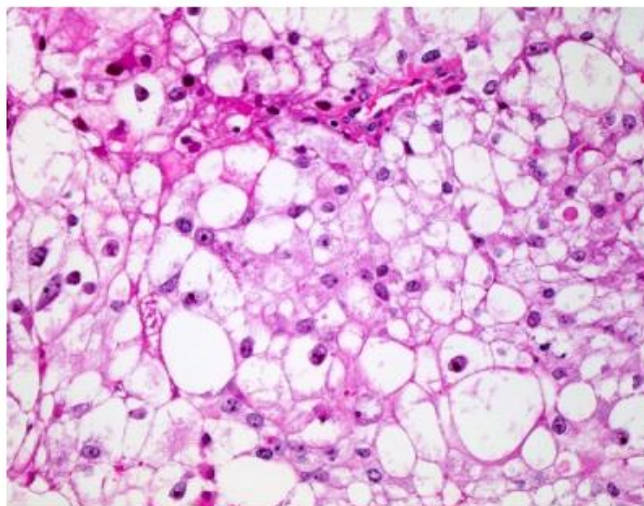


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Left: normal kidney. Right: 'clear-cell' pattern of renal cell carcinoma



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'Clear-cell' pattern of renal cell carcinoma - clear cytoplasm, small nuclei

*incidence of renal cell cancer is only slightly increased in patients with autosomal dominant polycystic kidney disease

Quick comparison: Carcinogens

Squamous cell carcinoma of the bladder cancer

- schistosomiasis

Transitional cell carcinoma

- aniline dyes

Relevant to my revision

Less relevant

Bladder cancer: risk factors

Risk factors for transitional cell carcinoma of the bladder include:

- Smoking
- Exposure to aniline dyes in the printing and textile industry: examples are 2-naphthylamine and benzidine
- Rubber manufacture
- Cyclophosphamide

Risk factors for squamous cell carcinoma of the bladder include:

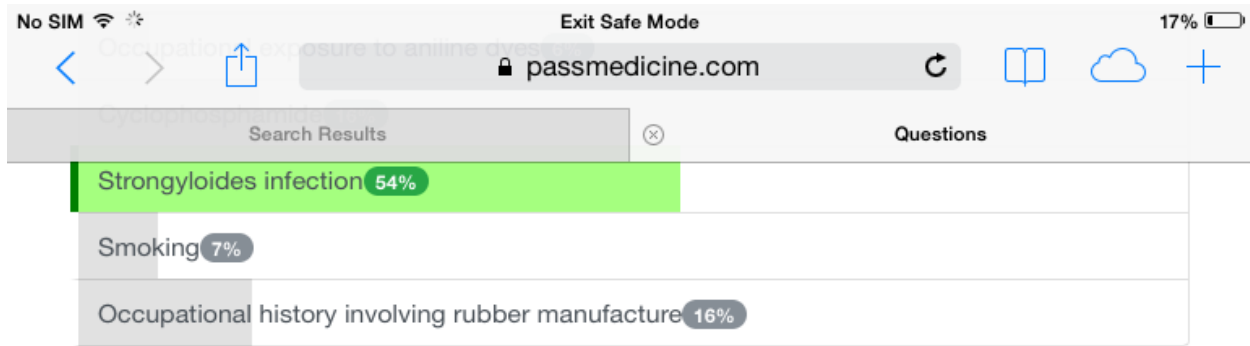
- Schistosomiasis
- Calmette-Guérin (BCG) treatment
- Smoking

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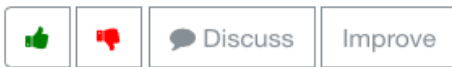
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Schistosomiasis rather than Strongyloides infection is associated with an increased risk of bladder cancer



Next question >

Bladder cancer: risk factors

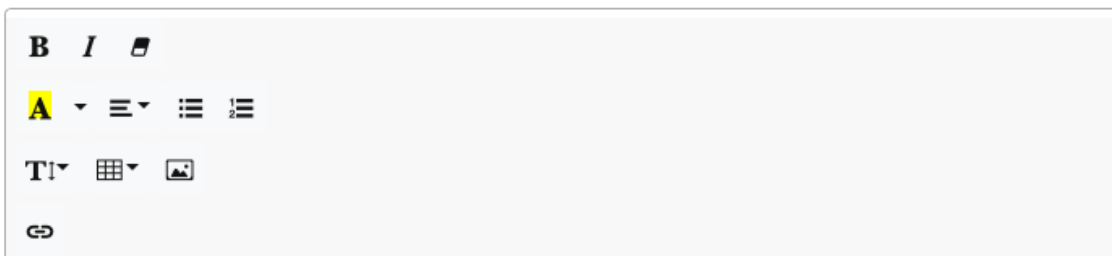
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Risk factors for squamous cell carcinoma of the bladder include:

- Schistosomiasis
- Calmette-Guérin (BCG) treatment
- Smoking

Next question >



Next question >

Prostate cancer: management

Localised prostate cancer (T1/T2)

Treatment depends on life expectancy and patient choice. Options include:

- conservative: active monitoring & watchful waiting
- radical prostatectomy
- radiotherapy: external beam and brachytherapy

Localised advanced prostate cancer (T3/T4)

Options include:

- hormonal therapy: see below
- radical prostatectomy
- radiotherapy: external beam and brachytherapy

Metastatic prostate cancer disease - hormonal therapy

Synthetic GnRH agonist

- e.g. Goserelin (Zoladex)
- cover initially with anti-androgen to prevent rise in testosterone

Anti-androgen

- cyproterone acetate prevents DHT binding from intracytoplasmic protein complexes

Orchidectomy

Next question >

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Prostate cancer: PSA testing

Prostate specific antigen (PSA) is a serine protease enzyme produced by normal and malignant prostate epithelial cells. It has become an important tumour marker but much controversy still exists regarding its usefulness as a screening tool.

The NHS Prostate Cancer Risk Management Programme (PCRMP) has published updated guidelines in 2009 on how to handle requests for PSA testing in asymptomatic men. A recent European trial (ERSPC) showed a statistically significant reduction in the rate of death prostate cancer by 20% in men aged 55 to 69 years but this was associated with a high risk of over-diagnosis and over-treatment. Having reviewed this and other data the National Screening Committee have decided not to introduce a prostate cancer screening programme yet but rather allow men to make an informed choice.

Age-adjusted upper limits for PSA were recommended by the PCRMP:

Age	PSA level (ng/ml)
50-59 years	3.0
60-69 years	4.0
> 70 years	5.0

PSA levels may also be raised by*:

- benign prostatic hyperplasia (BPH)
- prostatitis and urinary tract infection (NICE recommend to postpone the PSA test for at least 1 month after treatment)
- ejaculation (ideally not in the previous 48 hours)
- vigorous exercise (ideally not in the previous 48 hours)
- urinary retention
- instrumentation of the urinary tract

Poor specificity and sensitivity

- around 33% of men with a PSA of 4-10 ng/ml will be found to have prostate cancer. With a

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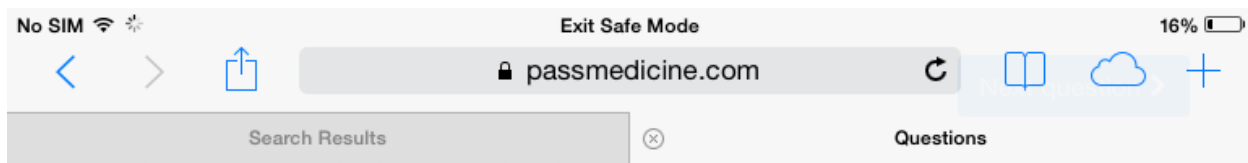
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- urinary retention
- instrumentation of the urinary tract

Poor specificity and sensitivity

- around 33% of men with a PSA of 4-10 ng/ml will be found to have prostate cancer. With a PSA of 10-20 ng/ml this rises to 60% of men
- around 20% with prostate cancer have a normal PSA
- various methods are used to try and add greater meaning to a PSA level including age-adjusted upper limits and monitoring change in PSA level with time (PSA velocity or PSA doubling time)

*whether digital rectal examination actually causes a rise in PSA levels is a matter of debate

Next question >



Benign prostatic hyperplasia

Benign prostatic hyperplasia (BPH) is a common condition seen in older men.

Risk factors

- age: around 50% of 50-year-old men will have evidence of BPH and 30% will have symptoms. Around 80% of 80-year-old men have evidence of BPH
- ethnicity: black > white > Asian

BPH typically presents with lower urinary tract symptoms (LUTS), which may be categorised into:

- voiding symptoms (obstructive): weak or intermittent urinary flow, straining, hesitancy, terminal dribbling and incomplete emptying
- storage symptoms (irritative) urgency, frequency, urgency incontinence and nocturia
- post-micturition: dribbling
- complications: urinary tract infection, retention, obstructive uropathy

Management options

- watchful waiting
- medication: alpha-1 antagonists, 5 alpha-reductase inhibitors. The use of combination therapy was supported by the Medical Therapy Of Prostatic Symptoms (MTOPS) trial
- surgery: transurethral resection of prostate (TURP)

Alpha-1 antagonists e.g. tamsulosin, alfuzosin

- decrease smooth muscle tone (prostate and bladder)
- considered first-line, improve symptoms in around 70% of men
- adverse effects: dizziness, postural hypotension, dry mouth, depression

5 alpha-reductase inhibitors e.g. finasteride

- block the conversion of testosterone to dihydrotestosterone (DHT), which is known to induce BPH
- unlike alpha-1 antagonists causes a reduction in prostate volume and hence may slow disease progression. This however takes time and symptoms may not improve for 6 months. They may also decrease PSA concentrations by up to 50%
- adverse effects: erectile dysfunction, reduced libido, ejaculation problems, gynaecomastia



Next question >

Epididymo-orchitis

Epididymo-orchitis describes an infection of the epididymis +/- testes resulting in pain and swelling. It is most commonly caused by local spread of infections from the genital tract (such as *Chlamydia trachomatis* and *Neisseria gonorrhoeae*) or the bladder.

The most important differential diagnosis is testicular torsion. This needs to be excluded urgently to prevent ischaemia of the testicle.

Features

- unilateral testicular pain and swelling
- urethral discharge may be present, but urethritis is often asymptomatic
- factors suggesting testicular torsion include patients < 20 years, severe pain and an acute onset

Management

- the British Association for Sexual Health and HIV (BASHH) produced guidelines in 2010
- if the organism is unknown BASHH recommend: ceftriaxone 500mg intramuscularly single dose, plus doxycycline 100mg by mouth twice daily for 10-14 days
- further investigations following treatment are recommended to exclude any underlying structural abnormalities

Next question >

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Scrotal problems



Epididymal cysts

Epididymal cysts are the most common cause of scrotal swellings seen in primary care.

Features

- separate from the body of the testicle
- found posterior to the testicle

Associated conditions

- polycystic kidney disease
- cystic fibrosis
- von Hippel-Lindau syndrome

Diagnosis may be confirmed by ultrasound.

Management is usually supportive but surgical removal or sclerotherapy may be attempted for larger or symptomatic cysts.

Hydrocele

A hydrocele describes the accumulation of fluid within the tunica vaginalis. They can be divided into communicating and non-communicating:

- communicating: caused by patency of the processus vaginalis allowing peritoneal fluid to drain down into the scrotum. Communicating hydroceles are common in newborn males (clinically apparent in 5-10%) and usually resolve within the first few months of life
- non-communicating: caused by excessive fluid production within the tunica vaginalis

Hydroceles may develop secondary to:

- epididymo-orchitis
- testicular torsion
- testicular tumours

Features

- soft, non-tender swelling of the hemi-scrotum. Usually anterior to and below the testicle
- the swelling is confined to the scrotum, you can get 'above' the mass on examination
- transilluminates with a pen torch
- the testis may be difficult to palpate if the hydrocele is large



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- transilluminates with a pen torch
- the testis may be difficult to palpate if the hydrocele is large

Diagnosis may be clinical but ultrasound is required if there is any doubt about the diagnosis or if the underlying testis cannot be palpated.

Management

- infantile hydroceles are generally repaired if they do not resolve spontaneously by the age of 1-2 years
- in adults a conservative approach may be taken depending on the severity of the presentation. Further investigation (e.g. ultrasound) is usually warranted however to exclude any underlying cause such as a tumour

Varicocele

A varicocele is an abnormal enlargement of the testicular veins. They are usually asymptomatic but may be important as they are associated with infertility.

Varicoceles are much more common on the left side (> 80%). Features:

- classically described as a 'bag of worms'
- subfertility

Diagnosis

- ultrasound with Doppler studies

Management

- usually conservative
- occasionally surgery is required if the patient is troubled by pain. There is ongoing debate regarding the effectiveness of surgery to treat infertility

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